The Effect of Menopause on the Periodontium- A Review

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Abstract

Sex hormones like estrogen and progesterone are responsible for physiological changes in women at specific phases of their life. Menopause is associated with symptoms of estrogen deficiency. Estradiol levels fall gradually in the years before menopause. Levels of follicle- stimulating hormone (FSH) and luteinizing hormone (LH) begin to rise and the levels of sex hormones begin to fluctuate. This causes changes in the periodontium like xerostomia, burning sensation in the oral mucosa, bleeding on probing and brushing, bad taste and alveolar bone loss. The most significant problem seen during menopause is osteoporosis. Osteoporosis is a disease characterized by low bone mass and fragility causing an increase in fracture risk. There is a reduction in bone mass caused by an imbalance between bone resorption and formation, favouring resorption resulting in demineralization of bone.

Keywords: Menopause; Osteoporosis; Sex hormones; Periodontal tissue

Introduction

The homeostasis of the periodontium involves complex multifactorial relationships, in which the endocrine system plays an important role[1]. Estrogen and Progesterone are responsible for physiological changes in women at specific phases of their life: puberty, menstrual cycle, pregnancy, menopause and post menopause [2].

Estrogen inhibits the expression of inflammatory cytokines important in bone resorption, and estrogen deficiency may contribute to more intense gingival inflammation during periodontitis and subsequent bone loss and may result in bone loss at both oral and skeletal sites. A number of studies have suggested that the risk of postmenopausal tooth loss is reduced by estrogen replacement [3]. Lower estrogen levels have been linked to gingival inflammation [4] and reduced clinical attachment levels [5]. In menopause, estrogen levels decline rapidly, which can lead to systemic bone loss [6].

The rate of bone loss in postmenopausal women predicts tooth loss for every 1%- per year decrease in whole body bone mineral density, the risk of tooth loss increases more than four times [7].

In fact, Kribbs [8] found that women with severe osteoporosis were three times more likely than healthy, age-matched controls to be edentulous (ie, to have fewer teeth). Famili et al. [9] found no association between systemic bone loss, periodontal disease and edentulism. This shows that the relationship between alveolar bone loss and systemic bone loss is multifactorial and not yet fully understood [10].

Nevertheless, the American Academy of Periodontology considers osteoporosis to be a risk factor for periodontal disease [11]. In fact, alveolar bone loss has been related not only to osteoporosis but also to osteopenia [12].

Factors Influencing Sex Hormone Effect on the Periodontium

Gender

Gender plays an important role in changes of the bone density throughout the entire skeleton. It is also known that women are much more affected than men (e.g. osteoporosis).

Lau et al. reported that 80% of the osteoporotic patients are female correlating with the higher frequency of hip fractures in females, who are also more likely to experience hormonal imbalance throughout their lives than males [13].

In addition, when the influence of gender on periodontal disease was studied, females were considered for several years to be more affected than males, although contradicting data have been reported. This disparity seems to be simply correlated with the fact that females are more likely to seek dental care than males [14].

Age

The biological changes on the periodontal tissues during different time points such as puberty, the menstrual cycle, pregnancy, menopause and oral contraceptive use have heightened interest in the relationship between steroid sex hormones and the health of the periodontium. Females seem to be more prone to hormone imbalance than males.

Puberty

Puberty is a complex process of sexual maturation resulting in an individual capable of reproduction [14,15]. It is also responsible for changes in physical appearance and behavior [14,16-18] that are related with increased levels of the steroid sex hormones, testosterone in males and estradiol in females. During puberty the production of sex hormones increases to a level that remains constant for the entire normal reproductive period. Changes in hormone levels have been related with an increased prevalence of gingivitis followed by remission [14], a situation that is not necessarily
associated with an increase in the amount of dental plaque [14,19]. The subgingival microflora is also altered during this period since the bacterial counts increase in number and there is a prevalence of certain bacterial species such as Prevotella intermedia and Capnocytophaga species [14,20,21].

Prevotella intermedia has been shown to possess the ability to substitute estrogen and progesterone for menodione (vitamin K) as an essential growth factor [22].

Capnocytophaga species, which often increase during puberty, have been associated with the increased bleeding tendency observed during this period of time [14,21].

Menopause and Postmenopause

In the premenopausal women, the principal circulating estrogen is 17b-estriadiol. As women approach menopause, the levels of estrogen begins to drop mainly during the late follicular and luteal phase of the menstrual cycle [23]. As a result of this physiologic situation, irregular cycles start to occur. Frequently the time frame between regular cycles and the cessation of menstrual periods called perimenopausal transition, is 2-7 years. During this period, the concentration of circulating estrogen decreases while follicle-stimulating hormone (FSH) and luteinizing hormone (LH) concentrations increase. Consequently the effects of estrogen are reduced, therefore compromising the anti-inflammatory effect of this hormone on the periodontium [14]. Progesterone is another sex hormone that may play an important role in bone metabolism during pre and post menopause [24].

It is believed that ovarian deficiency and associated alterations, but not aging are the predominant causes of bone loss during the first two decades after menopause.

Researchers have shown that progesterone may compete with glucocorticoids for an osteoblast receptor and inhibit the glucocorticoid induced osteoporosis. Therefore postmenopausal bone density reduction may be the result of a combination of inhibition of osteoclast down regulation by reduced estrogen and the increased cortisol inhibition of osteoblasts via the reduction of competition with progesterone [14].

Changes Seen in Periodontal Tissues Due to Menopause

Women appear to experience an increase in oral symptoms that may result from endocrine disturbances (reduced estrogen), calcium and vitamin deficiency and various psychologic factors during their menopausal years [25,26]. They may complain of dry mouth because of decreased salivary secretion as well as a burning sensation of the mouth and tongue. Taste sensation may change causing frequent complaints of a metallic taste [27]. Sex steroid hormone research has focused primarily on two cell groups the keratinocytes and the fibroblast [1]. During menopause women may experience dysesthesia, dental caries, periodontitis and an osteoporotic jawbone unsuitable for conventional dental devices and implants [28].

Osteoporosis is more common in women than men. Women are at a greater risk for osteoporosis after menopause [29] because estrogen levels decline rapidly, which may lead to systemic bone loss [30]. Bone turnover rate is higher in alveolar bone than long bones. Therefore, it was suggested that a systemic imbalance in bone resorption and deposition might be manifested earlier in the alveolar process than in other sites [31].

Kribbs reported that postmenopausal women with osteoporosis had decreased mandibular bone have long been considered to affect periodontal tissues and periodontal disease progression [32,33].

Treatment of periodontal disease has been primarily directed towards a microbiological etiology. Prevention of bone loss by modulating the host response to infection could be a new adjunctive method for the management of periodontitis [31]. Bisphosphonates, the most commonly prescribed therapy for osteoporosis, inhibit systemic bone resorption [34].

Drugs that alter bone metabolism, such as estrogen and bisphosphonates, were suggested by several case-control studies as a new approach to the treatment of periodontitis in post menopausal patients [31].

Serum osteocalcin is presently considered a valid marker of bone turnover when resorption and formation are coupled and a specific marker of bone formation when formation and resorption are uncoupled [35]. Bullon et al. reported that low serum osteocalcin concentration is associated with a significantly higher percentage of decrease in probing depth and clinical attachment level after periodontal treatment in postmenopausal women. Low saliva osteocalcin concentrations are significantly associated with a higher percentage of decrease in probing depth [36]. Lorne et al. reported the therapeutic potential of long term sub antimicrobial dose doxycycline therapy to reduce periodontal collagen breakdown and alveolar bone resorption in post menopausal women [37].

Periodontal infections can increase the systemic release of inflammatory cytokines, which accelerates systemic bone resorption. Indeed, vitamin D deficiency has been associated with a cytokine profile that favours greater inflammation (higher levels of C-reactive protein and interleukin 10) and vitamin D supplementation decreases circulating inflammatory markers. Therefore Luca et al. suggest that menopausal women should maintain an adequate vitamin D status in order to prevent and treat osteoporosis associated periodontal disease [38].

Sex hormones have been considered to affect periodontal tissues and periodontal disease progression [39,40]. Bharadwaj and colleagues reviewed the effect of menopause on the periodontium and reported that female sex hormones are neither necessary nor sufficient to produce gingival changes by themselves. However, they may alter periodontal tissue responses to microbial plaque and thus indirectly contribute to periodontal disease [41].

Clinical Significance

Buenacino and colleagues reviewed the association between menopause and periodontal disease and suggested that postmenopausal women can be managed, in part, by returning to the basics suggested by the ADA [42].

- Regular dental examinations; regular professional cleaning to remove bacterial plaque biofilm under the gum-line where a toothbrush will not reach.
- Daily oral hygiene practices to remove biofilm at and above the gum-line including brushing twice daily with ADA-accepted toothpaste.
- Replacing the toothbrush every 3–4 months (or sooner if the bristles begin to look frayed).
- Cleaning interproximally (between teeth) with floss or interdental cleaner.
• Maintaining a balanced diet.
• No smoking.

Conclusion

This review emphasizes on the effects of menopause on periodontal disease progression and wound healing. It is also clear that not all patients and their periodontium respond in the same way to similar amounts of circulating sexual hormones. The influence of sex hormones can be minimized with good plaque control as well as with hormonal replacement therapies.

Conflict of Interest

The authors deny any conflicts of interest related to the study.

References

